Prevalence of Risk Factors for Metabolic Syndrome in Adolescents

National Health and Nutrition Examination Survey (NHANES), 2001-2006

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Objective: To investigate the prevalence of distinct combinations of components of the metabolic syndrome among adolescents.

Design: A complex, multistage, stratified geographic area design for collecting representative data from the non-institutionalized US population.

Setting: The NHANES, an ongoing surveillance of the nation’s health conducted by the Centers for Disease Control and Prevention.

Participants: Two thousand four hundred fifty-six Hispanic, white, and black adolescents aged 12 to 19 years observed in the 2001-2002, 2003-2004, and 2005-2006 NHANES data releases.

Main Outcome Measures: Metabolic syndrome was defined as having 3 or more disorders in the following measurements: waist circumference, blood pressure, fasting triglycerides, high-density lipoprotein serum cholesterol, and glucose.

Results: About half of the participants had at least 1 disordered measurement, with an overall metabolic syndrome prevalence of 8.6% (95% confidence interval, 6.5%-10.6%). Prevalence was higher in males (10.8%) than females (6.1%), and in Hispanic (11.2%) and white (8.9%) individuals than in black individuals (4.0%). In black females, there was a high prevalence of a large waist circumference (23.3%), but no component of metabolic syndrome dominated its diagnosis in black adolescents of either sex. A large waist circumference and high fasting triglyceride and low high-density lipoprotein serum cholesterol concentrations were salient factors in Hispanic and white adolescents of both sexes; high glucose concentrations were prominent among Hispanic and white males.

Conclusion: The low prevalence of metabolic syndrome in black adolescents, in parallel with uniformly low prevalence of all 5 risk factors among those with metabolic syndrome, portend ethnic disparities in the time table for early onset of cardiometabolic disorders.


Prevalence trends for obesity, type 2 diabetes mellitus, and other obesity-related disorders in the United States and many other countries are disturbing. Obesity’s role in the series of concurrent cardiometabolic changes that often precede more serious cardiovascular disorders is being recognized with increasing urgency. Obesity, especially abdominal and central adiposity, as measured herein by waist circumference (WC), is often linked to insulin resistance and other diagnostic correlates of metabolic syndrome: elevated triglyceride concentrations, low high-density lipoprotein cholesterol (HDL-C) concentrations, elevated blood pressure (BP), and elevated fasting glucose concentrations. These 5 factors appear to be closely allied with increased risk of developing type 2 diabetes mellitus and cardiovascular diseases. When a person is found to have any 3 of the 5 component risk factors, concern grows, warranting a diagnosis of metabolic syndrome (MetS). Although conflicting opinions have been reported concerning the diagnostic value of MetS, the concept provides a readily measured set of criteria that could lead to more intense clinical focus on the underlying causes and thereby reinforces the utility of lifestyle changes and perhaps additional clinical interventions. Our argument is that several cardiometabolic risk factors within 1 individual, even at subclinical levels that would trigger traditional treatment options, predict future progression to overt chronic disease. Thus, MetS may be a sentinel marker for highlighting the need for more aggressive intervention efforts beyond the traditional treatment options for overweight and obese youths.

According to recent estimates, MetS affects, depending on the definition, 21% to

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38.9% of the US adult population overall; the prevalence in adults aged 20 to 29 years was found to be between 9.5% and 19.6%. Many authors have reported adolescent prevalence data for MetS using various diagnostic criteria. Although agreement is lacking on criteria for diagnosing MetS in adolescents, the diagnostic criteria typically involve the same 5 risk factors identified in adults, with modifications to the cut-off values for defining disorders in specific components. The cut-off values give rise to conflicting definitions and prevalences of MetS in adolescents. Cook et al investigated 4 previously reported definitions of adolescent MetS that included application of adult cutoffs in addition to 3 sets of modified cutoffs. These definitions led to disparate estimates, ranging from 2.0% to 9.4%, for the prevalence of adolescent MetS. As shown in Figure 1, other studies have also reported instabilities in prevalence estimates as different diagnostic criteria are invoked.

The cardiovascular risk related to MetS appears to travel from childhood to young adulthood. A better understanding of the determinants of MetS during adolescence might provide insights into preventive interventions for improving health outcomes during adolescence and reducing the incidence of cardiovascular disease in adults. The aim of this study was to investigate the prevalence of different combinations of the 5 component risk factors for MetS in search of the most influential diagnostic determinants among adolescents aged 12 to 19 years in the United States. A second objective was to determine differences in overall prevalence of MetS in 6 sex-ethnicity subgroups.

**DATA FOR DIAGNOSING MetS**

Participants were required to come to the mobile examination clinic before 9 AM after fasting for at least 9 hours. If they arrived having fasted for less than 8.5 hours, they were assigned a sampling weight equal to 0 as part of the NHANES protocol. This reduced the sample size for our analysis by 204 participants. Waist circumference was measured to the nearest 0.1 cm at minimal respiration at the end of normal expiration with a steel measuring tape placed at the high point of the iliac crest when the participant was in a standing position. Diastolic and systolic BP measurements were obtained using replicated measurements. After resting quietly in a sitting position for 5 minutes and determination of the maximum inflation level, up to 4 consecutive BP readings were obtained with a mercury manometer. Mean values of replicate systolic and diastolic measurements provided estimates of current BP levels. Glucose concentration was determined by a hexokinase method, triglyceride concentration was measured enzymatically using a series of coupled reactions, and HDL-C concentration was measured directly. Unfortunately, the Centers for Disease Control and Prevention have issued an advisory regarding making inferences from HDL-C data in 2003-2006 NHANES releases, warning that specific measurements may be overestimated by a mean of 3 mg/dL. (to convert to millimoles per liter, multiply by 0.0259).

**DIAGNOSTIC CRITERIA FOR MetS**

In this communication, adolescents were classified as having MetS if they had any 3 of the following: a WC in the 90th percentile for their age and sex according to 1988-1994 NHANES III data; either systolic or diastolic BP in the 90th percentile for their height, age, and sex as previously specified; triglyceride concentration of 110 mg/dL or greater (to convert to millimoles per liter, multiply by 0.0113); HDL-C concentration of 40 mg/dL or less; and glucose concentration of 100 mg/dL or greater (to convert to millimoles per liter, multiply by 0.0555). These criteria are the same as those used by Ford et al.
STATISTICAL ANALYSIS

All analyses were performed using procedures for sample survey data that are readily available in SAS, version 9.1 (SAS Institute, Cary, North Carolina). Prevalence data were reported for MetS and disorders (elevated or low measurements) in the 5 component variables used to classify adolescents with respect to their MetS status. Estimates of the number of adolescents in various subpopulations were also reported. Prevalence data expressed as a percentage with 95% confidence intervals (CIs), weighted to be nationally representative, were compiled for the overall sample, males and females, ethnicity groups, and within sex-ethnicity subgroups. Ethnicity was categorized as Hispanic, white, or black. Overall and sex-specific time trends were evaluated during the periods 2001-2002, 2003-2004, and 2005-2006.

RESULTS

SAMPLE DESCRIPTION

There were 2456 adolescents in the study sample, which represented a population of almost 29 million adolescents (Table 1). Slightly more than half of the weighted sample were males (51.6%); most were white (68%); 16.9% were Hispanic; and 15.1% were black. There were 11.2% to 13.1% in each age group (12-19 years).

PREVALENCE OF MetS

An estimated 8.6% of adolescents in the study had MetS (95% CI, 6.5%-10.6%), which extrapolates to almost 2.5 million adolescents in the general population (Table 2). The prevalence of MetS was highest in Hispanic males (odds ratio [OR], 3.69; 95% CI, 2.05-6.65), followed by white males (OR, 3.33; 95% CI, 1.61-6.88); Hispanic females (OR, 2.58; 95% CI, 1.20-5.54), white females (OR, 1.52; 95% CI, 0.75-3.09), and black females (OR, 1.09; 95% CI, 0.47-2.56) (white females were the reference). Prevalence estimates throughout the 2-year period did not reveal clear trends.

PREVALENCE OF INDIVIDUAL RISK FACTORS

Overall, 19.1% (95% CI, 16.2%-22.0%) of the adolescents had excess central adiposity (large WC). In black adolescents, large WCs were more prevalent in females than males (23.3% vs 11.9%). Among males, large WCs were more prevalent in Hispanic (21.5%) and white (18.4%) adolescents compared with black adolescents (11.9%). Almost 2 million adolescents (6.9%; 95% CI, 5.1%-8.6%) were calculated to have elevated BP. Among males, the prevalence of high BP was lower in Hispanic adolescents (4.9%) than in black adolescents (8.3%), whereas among females, prevalence was lowest for white adolescents (6.4%). Overall, a high triglyceride concentration was the most prevalent disorder (25.6%). The prevalence was significantly greater in Hispanic (26.1%) and white (28.9%) adolescents than in black adolescents (9.8%). Ethnicity prevalence differences in males were similar to those in females, corresponding to overall disparities. About nineteen percent (19.3%) of sampled adolescents had a low HDL-C concentration; prevalence was significantly greater among males (24.6%) than in females (13.7%) and in Hispanic (19.9%) and white (21.1%) adolescents than in black adolescents (10.6%). Ethnicity prevalence differences in males were similar to overall disparities, whereas in females, the prevalence was lower but ethnic disparities were within the range of sampling variability. We estimated that more than 4 million adolescents (14.0%) have an elevated glucose concentration. Prevalence was greater among males than females (19.8% vs 7.9%) and in Hispanic (15.2%) and white (14.8%) compared with black (9.4%) adolescents. Ethnic differences within sex were similar to corresponding overall disparities but were significant only in Hispanic compared with black males (22.4% vs 13.5%).

PREVALENCE OF RISK FACTOR COMBINATIONS

About half of the adolescents had disorders for 1 or more components of MetS, 42% had disorders for 1 or 2 components, and 8.6% had 3 or more disorders. More males than females had at least 1 disorder (55.5% vs 45.5%); 3.2% of males compared with 2.0% of females had 4 or more disorders. Fewer adolescents were found to have 3 or more disorders in 2003-2004, in concordance with the findings summarized in Table 3. This appears to be a result of fewer males having 3 disordered components and more males having 2 (pre-MetS) components in addition to fewer females having 3 and more having 0 disordered components in 2003-2004.

Prevalence estimates for distinct combinations of diagnostic components for MetS are presented in Table 3. The prevalence of only 1 disordered component among males was highest for high triglyceride concentration (8.8%) followed by high glucose concentration (8.3%). In females, it was highest for triglyceride concentration (9.7%) followed by high WC (8.1%). The prevalence of

Table 1. Characteristics of Study Sample and Estimated US Adolescent Population Aged 12 to 19 Years

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Study Sample</th>
<th>Population, ×1000</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>2456</td>
<td>28 729</td>
<td>2.5</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>1288</td>
<td>14 810</td>
<td>51.6</td>
</tr>
<tr>
<td>F</td>
<td>1168</td>
<td>13 918</td>
<td>48.4</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>899</td>
<td>4868</td>
<td>16.9</td>
</tr>
<tr>
<td>White</td>
<td>715</td>
<td>19 528</td>
<td>68.0</td>
</tr>
<tr>
<td>Black</td>
<td>842</td>
<td>4333</td>
<td>15.1</td>
</tr>
<tr>
<td>Age, y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>310</td>
<td>3500</td>
<td>12.2</td>
</tr>
<tr>
<td>13</td>
<td>325</td>
<td>3762</td>
<td>13.1</td>
</tr>
<tr>
<td>14</td>
<td>292</td>
<td>3449</td>
<td>12.0</td>
</tr>
<tr>
<td>15</td>
<td>297</td>
<td>3716</td>
<td>12.9</td>
</tr>
<tr>
<td>16</td>
<td>318</td>
<td>3742</td>
<td>13.0</td>
</tr>
<tr>
<td>17</td>
<td>314</td>
<td>3655</td>
<td>12.7</td>
</tr>
<tr>
<td>18</td>
<td>315</td>
<td>3694</td>
<td>12.9</td>
</tr>
<tr>
<td>19</td>
<td>285</td>
<td>3209</td>
<td>11.2</td>
</tr>
</tbody>
</table>
a distinct combination of 2 risk factors among males was highest for high triglyceride and HDL-C concentrations (4.9%) followed by high triglyceride and glucose concentrations (2.6%); in females, it was highest for high WC and triglyceride concentration (3.8%) followed by high triglyceride and low HDL-C concentrations (2.6%). The highest prevalence for 3 distinct components was high WC, high triglyceride concentration, and low HDL-C concentration for both males (3.7%) and females (2.1%).

Among the 10.8% of males with MetS, 34.3% had disorders for WC, triglycerides, and HDL-C; in females, the comparable value was 34.4%. An estimated 2.2% of males vs 0.9% of females had simultaneous disorders for WC and triglyceride, HDL-C, and glucose concentrations; among those with MetS, this figure rises to 20.4% and 14.8% in males and females, respectively.

**PREVALENCE OF INDIVIDUAL DISORDERS IN ADOLESCENTS WITH MetS**

With examination of the prevalence of individual components of MetS in adolescents who have any distinct combination of 3 or more components, it appears that elevated BP does not play a primary role in early onset of this syndrome (Figure 2). Among Hispanic and white adolescents, prevalence is prominent for disorders in WC, triglycerides, and HDL-C, which seem to be the most influential contributors to the diagnosis of MetS in both sexes, with glucose concentration having some degree of importance in males. Although black females demonstrated a moderately high prevalence of central adiposity, a leading driver of MetS was not found in black adolescents.

**COMMENT**

Our findings affirm others’ conclusion that the prevalence of impaired biomarkers for early-onset metabolic abnormalities represents a legitimate health concern. The reported prevalence of MetS in adolescents living in the United States has varied widely from study to study (10%-21%). At least some of this variability is attributable to the use of different criteria in defining MetS in adoles-

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**Table 2. Population Prevalence of Metabolic Syndrome and Its Risk Factors in US Adolescents Aged 12 to 19 Years**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Overall</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic syndrome, ≥3 of 5 criteria</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>11.2 (8.0-14.5)</td>
<td>12.9 (9.3-16.6)</td>
<td>9.4 (4.7-14.1)</td>
</tr>
<tr>
<td>White</td>
<td>8.9 (6.1-11.7)</td>
<td>11.8 (7.3-16.4)</td>
<td>5.8 (3.6-7.9)</td>
</tr>
<tr>
<td>Black</td>
<td>4.0 (2.7-5.4)</td>
<td>3.9 (1.8-5.9)</td>
<td>4.2 (1.9-6.5)</td>
</tr>
<tr>
<td>Total</td>
<td>8.6 (6.5-10.6)</td>
<td>10.8 (7.3-14.3)</td>
<td>6.1 (4.6-7.7)</td>
</tr>
<tr>
<td>NHANES period</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2001-2002</td>
<td>9.0 (5.2-12.7)</td>
<td>12.5 (5.0-20.0)</td>
<td>5.5 (2.4-8.5)</td>
</tr>
<tr>
<td>2003-2004</td>
<td>6.5 (4.1-9.0)</td>
<td>8.6 (4.5-12.8)</td>
<td>4.2 (1.7-6.8)</td>
</tr>
<tr>
<td>2005-2006</td>
<td>10.1 (5.3-14.9)</td>
<td>11.5 (4.2-18.8)</td>
<td>8.6 (5.7-11.6)</td>
</tr>
<tr>
<td>Waist circumference, ≥90th percentile</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>22.7 (18.6-26.7)</td>
<td>21.5 (17.0-26.0)</td>
<td>23.9 (19.0-28.8)</td>
</tr>
<tr>
<td>White</td>
<td>18.5 (14.8-22.2)</td>
<td>18.4 (14.4-22.5)</td>
<td>18.6 (13.4-23.9)</td>
</tr>
<tr>
<td>Black</td>
<td>17.5 (14.6-20.4)</td>
<td>11.9 (9.1-14.7)</td>
<td>23.3 (18.8-27.8)</td>
</tr>
<tr>
<td>Total</td>
<td>19.1 (16.2-22.0)</td>
<td>18.0 (14.6-21.4)</td>
<td>20.2 (16.0-24.5)</td>
</tr>
<tr>
<td>Blood pressure, ≥90th percentile</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>6.9 (3.5-10.2)</td>
<td>4.9 (2.7-7.0)</td>
<td>9.0 (3.7-14.3)</td>
</tr>
<tr>
<td>White</td>
<td>6.3 (4.2-8.5)</td>
<td>6.3 (3.8-8.8)</td>
<td>6.4 (3.4-9.4)</td>
</tr>
<tr>
<td>Black</td>
<td>9.1 (7.5-10.8)</td>
<td>8.3 (6.0-10.5)</td>
<td>10.1 (7.5-12.6)</td>
</tr>
<tr>
<td>Total</td>
<td>6.9 (5.1-8.6)</td>
<td>6.3 (4.0-8.7)</td>
<td>7.4 (4.9-10.0)</td>
</tr>
<tr>
<td>Triglycerides, ≥10 mg/dL</td>
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<td></td>
<td></td>
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<tr>
<td>Hispanic</td>
<td>26.1 (22.0-30.1)</td>
<td>27.5 (23.1-31.9)</td>
<td>24.6 (19.1-30.1)</td>
</tr>
<tr>
<td>White</td>
<td>28.9 (24.7-33.2)</td>
<td>31.5 (24.8-38.2)</td>
<td>26.2 (21.3-31.0)</td>
</tr>
<tr>
<td>Black</td>
<td>9.8 (8.0-11.6)</td>
<td>10.2 (7.7-12.7)</td>
<td>9.4 (6.9-11.9)</td>
</tr>
<tr>
<td>Total</td>
<td>25.6 (22.4-28.8)</td>
<td>27.7 (22.8-32.5)</td>
<td>23.3 (19.7-27.0)</td>
</tr>
<tr>
<td>HDL-C, ≤40 mg/dL</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>19.9 (16.5-23.4)</td>
<td>24.0 (20.0-27.9)</td>
<td>15.6 (9.7-21.6)</td>
</tr>
<tr>
<td>White</td>
<td>21.1 (18.1-24.1)</td>
<td>27.9 (23.3-32.4)</td>
<td>13.9 (10.2-17.6)</td>
</tr>
<tr>
<td>Black</td>
<td>10.6 (8.2-12.9)</td>
<td>10.8 (7.5-13.7)</td>
<td>10.6 (7.1-14.1)</td>
</tr>
<tr>
<td>Total</td>
<td>19.3 (17.1-21.6)</td>
<td>24.6 (21.2-28.1)</td>
<td>13.7 (10.7-16.6)</td>
</tr>
<tr>
<td>Fasting glucose, ≥100 mg/dL</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>15.2 (11.7-18.6)</td>
<td>22.4 (17.6-27.2)</td>
<td>7.4 (5.2-9.7)</td>
</tr>
<tr>
<td>White</td>
<td>14.8 (11.1-18.5)</td>
<td>20.6 (15.3-25.8)</td>
<td>8.6 (5.8-11.4)</td>
</tr>
<tr>
<td>Black</td>
<td>9.4 (6.9-11.9)</td>
<td>13.5 (9.6-17.3)</td>
<td>5.2 (2.4-8.1)</td>
</tr>
<tr>
<td>Total</td>
<td>14.0 (11.4-16.6)</td>
<td>19.8 (16.2-23.5)</td>
<td>7.9 (5.8-9.9)</td>
</tr>
</tbody>
</table>

Abbreviations: HDL-C, high-density lipoprotein cholesterol; NHANES, National Health and Nutrition Examination Survey.

SI conversion factors: To convert fasting glucose to millimoles per liter, multiply by 0.0555; HDL-C to millimoles per liter, multiply by 0.0259; and triglycerides to millimoles per liter, multiply by 0.0113.

Sample weights were used to calculate population estimates.
Prevalence of central adiposity was lowest in black adolescents (11.2%), followed by white (8.9%) and Hispanic (4.0%) adolescents. In white adolescents, prevalence of 8.6% is in the upper tertile of estimates from other NHANES data (2001-2006), our estimated overall prevalence was significantly higher in males than females. There are some indications in Figure 1 as well as in Table 2 that prevalence of MetS in adolescents is increasing with time, but the evidence is not conclusive. Prevalence of central adiposity was lowest in black males at 11.9%, but it exceeded 18.4% in all other sex-ethnic groups. It was highest in Hispanic females (23.9%) but relatively low in black adolescents. Low HDL-C was highly prevalent in both sexes of Hispanic and white adolescents compared with black adolescents. Prevalence of elevated glucose concentration was high in Hispanic and white males, intermediate in black males, and relatively low in all females. Prevalence of elevated BP was moderately low in all sex-ethnicity groups.

Table 3. Patterns of Disordered Diagnostic Components for Metabolic Syndrome in US Adolescents in the NHANES, 2001-2006^a^

<table>
<thead>
<tr>
<th>MetS Component</th>
<th>Study Participants, No. (%)</th>
<th>Overall (n=2456)</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>1297 (49.3)</td>
<td>640 (44.5)</td>
<td>657 (54.5)</td>
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<tr>
<td>WC</td>
<td>163 (5.9)</td>
<td>55 (3.8)</td>
<td>108 (8.1)</td>
<td></td>
</tr>
<tr>
<td>BP</td>
<td>60 (1.8)</td>
<td>30 (1.8)</td>
<td>30 (1.8)</td>
<td></td>
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<tr>
<td>HDL-C</td>
<td>123 (5.1)</td>
<td>77 (6.5)</td>
<td>46 (3.6)</td>
<td></td>
</tr>
<tr>
<td>GLU</td>
<td>150 (6.1)</td>
<td>110 (8.3)</td>
<td>40 (3.6)</td>
<td></td>
</tr>
<tr>
<td>WC + BP</td>
<td>20 (0.8)</td>
<td>7 (0.4)</td>
<td>13 (1.0)</td>
<td></td>
</tr>
<tr>
<td>WC + TG</td>
<td>63 (2.7)</td>
<td>23 (1.7)</td>
<td>40 (3.8)</td>
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</tr>
<tr>
<td>WC + HDL-C</td>
<td>38 (1.5)</td>
<td>18 (2.4)</td>
<td>20 (1.4)</td>
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<tr>
<td>WC + GLU</td>
<td>24 (0.7)</td>
<td>15 (0.9)</td>
<td>9 (0.5)</td>
<td></td>
</tr>
<tr>
<td>BP + TG</td>
<td>11 (0.4)</td>
<td>4 (0.1)</td>
<td>7 (1.2)</td>
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</tr>
<tr>
<td>BP + HDL-C</td>
<td>10 (0.4)</td>
<td>5 (0.7)</td>
<td>5 (0.7)</td>
<td></td>
</tr>
<tr>
<td>BP + GLU</td>
<td>6 (0.4)</td>
<td>4 (0.5)</td>
<td>2 (0.3)</td>
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<tr>
<td>TG + HDL-C</td>
<td>67 (2.7)</td>
<td>44 (4.9)</td>
<td>23 (2.6)</td>
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<tr>
<td>TG + GLU</td>
<td>36 (1.4)</td>
<td>27 (2.6)</td>
<td>9 (0.8)</td>
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</tr>
<tr>
<td>HDL-C + GLU</td>
<td>28 (0.8)</td>
<td>25 (1.4)</td>
<td>3 (0.2)</td>
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</tr>
<tr>
<td>WC + BP + TG</td>
<td>8 (0.3)</td>
<td>2 (0.3)</td>
<td>6 (0.4)</td>
<td></td>
</tr>
<tr>
<td>WC + BP + HDL-C</td>
<td>9 (0.3)</td>
<td>4 (0.4)</td>
<td>5 (0.3)</td>
<td></td>
</tr>
<tr>
<td>WC + BP + GLU</td>
<td>5 (0.2)</td>
<td>1 (&lt;0.1)</td>
<td>4 (0.4)</td>
<td></td>
</tr>
<tr>
<td>WC + TG + HDL-C</td>
<td>65 (2.9)</td>
<td>38 (3.7)</td>
<td>27 (2.1)</td>
<td></td>
</tr>
<tr>
<td>WC + TG + GLU</td>
<td>16 (0.5)</td>
<td>10 (0.7)</td>
<td>6 (0.2)</td>
<td></td>
</tr>
<tr>
<td>WC + HDL-C + GLU</td>
<td>6 (0.3)</td>
<td>6 (0.6)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>BP + TG + HDL-C</td>
<td>5 (0.3)</td>
<td>3 (0.3)</td>
<td>2 (0.3)</td>
<td></td>
</tr>
<tr>
<td>BP + TG + GLU</td>
<td>5 (0.4)</td>
<td>5 (0.7)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>BP + HDL-C + GLU</td>
<td>2 (0.1)</td>
<td>1 (0.1)</td>
<td>1 (&lt;0.1)</td>
<td></td>
</tr>
<tr>
<td>TG + HDL-C + GLU</td>
<td>13 (0.6)</td>
<td>9 (0.7)</td>
<td>4 (0.5)</td>
<td></td>
</tr>
<tr>
<td>WC + BP + TG + HDL-C</td>
<td>5 (0.3)</td>
<td>1 (&lt;0.1)</td>
<td>4 (0.6)</td>
<td></td>
</tr>
<tr>
<td>WC + BP + TG + GLU</td>
<td>5 (0.2)</td>
<td>3 (0.3)</td>
<td>2 (&lt;0.1)</td>
<td></td>
</tr>
<tr>
<td>WC + BP + HDL-C + GLU</td>
<td>3 (0.1)</td>
<td>2 (0.1)</td>
<td>1 (0.1)</td>
<td></td>
</tr>
<tr>
<td>WC + TG + HDL-C + GLU</td>
<td>34 (1.6)</td>
<td>27 (2.2)</td>
<td>7 (0.9)</td>
<td></td>
</tr>
<tr>
<td>BP + WC + TG + HDL-C + GLU</td>
<td>8 (0.3)</td>
<td>6 (0.7)</td>
<td>2 (0.3)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: BP, blood pressure; GLU, glucose; HDL-C, high-density lipoprotein cholesterol; NHANES, National Health and Nutrition Examination Survey; TG, triglyceride; WC, waist circumference.

^a^ Sample weights were used to calculate population estimates.

^b^ Waist circumference in the 90th percentile; BP in the 90th percentile; TG, 110 mg/dL or greater (to convert to millimoles per liter, multiply by 0.0113); HDL-C, 40 mg/dL or less (to convert to millimoles per liter, multiply by 0.0259); and GLU, 100 mg/dL or greater (to convert to millimoles per liter, multiply by 0.0555).
centration was not. However, prevalence of disordered
WC, triglyceride concentration, and HDL-C concentra-
tion was only moderately high in white females. Al-
though WC was slightly elevated in black adolescents,
one of the individual components of MetS stood out sig-
nificantly as determinants of the syndrome. Overall, BP
made only a minor contribution to MetS; this result is
consistent with results of a previous study in European,
African, and Cuban American adults aged 25 to 44 years
in whom insulin resistance, obesity, and lipid factors most
strongly represented MetS.30 We observed a low preva-
ence of MetS in black adolescents of both sexes, but preva-
ience of large WCs stood out only slightly in females, and
overall we did not find a pattern of WC or any other spe-
cific components driving the diagnosis of MetS.

Pediatric MetS has been reported to predict adult MetS
and type 2 diabetes mellitus.19 In adults, as in adoles-
cents, the epidemiology of MetS is unclear because the
diagnostic definition lacks consensus of scientific opin-
tions; hence, the implications of disparities among preva-
ience estimates have often been conflicting.9 We found
prevalence to be highest in Hispanic adolescents (males
higher than females) followed by white adolescents (males
lower than females) and black adolescents (males only slightly lower than females). This rela-
tively high prevalence of MetS in Hispanic and white ado-
lescents and the conflicting low prevalence in black adoles-
cents has been recently observed by others.10,12 Al-
though the low prevalence of MetS among black indi-
viduals during adolescence would be expected to trans-
late to a subsequent lower risk of developing comorbidities, prevalence has been found to be high in
black compared with white adults for hypertension, dia-
betes, and cardiovascular disease.34 The prevalence of MetS
was found to be very high among adult black partici-
ants in the Jackson Heart Study; moreover, elevated BP,
large WC, and low HDL-C were each highly prevalent
among those with MetS.32

A natural hypothesis is that the sequence of events lead-
ing to definitive MetS begins with the development of obe-
sity and/or insulin resistance then continues by taking
many alternate pathways until metabolic irregularities
evolve into life-threatening diseases. We found that many
adolescents were not obese and yet had impairments in
1 or more of the following 4 components: BP, triglycer-ides, HDL-C, and glucose (Table 3). That many adoles-
cents develop other metabolic disorders before they be-
come centrally obese suggests that there may be additional
origins of MetS. On the other hand, our definition of large
WC may be too conservative in relation to identifying
risk of metabolic disorder, so that overweight adoles-
cents who are below our cut-off point for central obe-
sity may be at risk for impaired triglyceride, HDL-C, and

glucose concentrations. Also, some adolescents may be
obese overall without being centrally obese. However, the
high correlation between body mass index and WC found
in our investigation as well as in a previous study ($r=0.94$
and $r=0.93$, respectively) does not support this hypo-
thesis.33 Using the 95th percentile for body mass index as
the threshold criterion for obesity, we found the overall
prevalence of MetS to be 8.3% (a value that is compa-
rable with the prevalence of high WC [8.6%]). Using body

mass index rather than WC as the criterion for deter-
mining obesity, we observed that the revised prevalence
was 12.5% (vs 12.9%) in male and 8.8% (vs 9.4%) in fe-
male Hispanic adolescents; 12.0% (vs 11.8%) in male and
5.1% (vs 5.8%) in female white adolescents; and 4.2%
(vs 3.9%) in male and 4.2% (vs 4.2%) in female black
adolescents. There is evidence that additional cardio-
metabolic risk due to obesity in adults is minimal once
the risk attributable to having an excessively large WC
is accounted for.34 Furthermore, poor cardiorespiratory
fitness may place many at metabolic risk,35 and fitness
can play a role even in those who are not obese.

The cross-sectional data in this NHANES study pro-
hibit a direct calculation of disease incidence and there-
fore we cannot make direct comparisons among sub-
groups regarding risk of developing MetS. Because specific
measurements of HDL-C may have been overestimated
in 2003-2006 NHANES releases and in light of the fact
that low concentrations of HDL-C are in the direction
of impairment, the prevalence data for HDL-C and MetS
may be underestimated in our analysis. The data also limit
our ability to investigate the time in which specific indi-
viduals develop disorders in the criteria used to clas-
sify adolescents with respect to MetS prevalence. Fur-
thermore, despite the large samples overall, sampling
variability becomes large and the stability of estimates is
compromised, as the sample sizes decrease when numer-
ous subgroups are investigated. As a result, some ob-
served differences among sex-ethnic subgroups may be
large, yet within the range of sampling variability and other
subgroup differences, such as those among sex-ethnic sub-
groups with respect to different component combina-
tions, were not investigated.

The American Heart Association recommends that
treatment options for childhood obesity be based on the
severity of obesity and the presence or absence of co-
morbidities.36 In the present study, 5.9% of the adoles-
cents had an elevated WC in the absence of other com-
ponent risk factors (Table 3). However, an additional 7.2%
of the adolescents had an elevated WC in the presence
of MetS. Of the 472 participants who had an elevated WC,
164 (35%) had other component risk factors to make the
diagnosis of MetS. Of the 25 study participants who had
MetS but did not have an elevated WC, 20 had an el-
vated glucose concentration as 1 of their 3 disorders,
whereas the other 5 had elevated BP and triglyceride con-
centrations and low HDL-C. Although each component
risk factor may be managed separately, it would be pru-
dent to identify those with multiple disorders, irrespec-
tive of obesity, and provide them with more aggressive
treatment and management options.

This work clearly identifies a different timetable and
perhaps a different pathway for developing MetS in black
adolescents. Although the prevalence of central adiposity
remained substantially lower in black males than in
other sex-ethnicity groups throughout adolescence, it was
very high in black females, but males and females both
had a relatively low prevalence of MetS. It is possible
that black individuals have a propensity for developing clus-
ters of component disorders later in adolescence and,
hence, demonstrate a lower prevalence of metabolic dis-
orders despite greater proclivity for cardiometabolic dis-


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eases in early adulthood compared with Hispanic and white individuals. The relatively low prevalence of all of the individual components among black individuals, except for the elevated prevalence of large WC in females, is consistent with a delayed onset, but a complete explanation of the ethnic disparities requires further study.

Accepted for Publication: November 21, 2008.
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Financial Disclosure: None reported.

Funding/Support: This research was supported by the Pennington Biomedical Research Center. Dr Katzmarzyk is supported in part as the Louisiana Public Facilities Authority Endowed Chair in Nutrition, and Dr Bouchard is funded in part as the George A. Bray Jr Chair in Nutrition.

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